Where exsanguination is great, nothing can replace transfusion; nevertheless, it will be well to remember the use of radium as an emergency measure. It is easily applied and, in the dosages Hoffman administers, is without danger.

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Artificial Diphtheria Antitoxins.—Traditional immunology pictures specific antitoxins as preformed, hereditary, specific intracellular antidotes, quantitatively increased and liberated into the blood stream as a result of toxic assault. With this conception of antibody formation, a test-tube synthesis of diphtheria antitoxin is inconceivable, without previous detailed knowledge of its exact chemical composition.

The newer immunology,[1] in contrast, pictures antitoxins, not as preformed hereditary antidotes that need but to be quantitatively increased to give immunity, but as absolutely new biochemical complexes resulting from chemical interaction between toxins and normal humoral or cellular proteins. Or, in the words of some of the newer immunologists, they are nonspecific hereditary defenses metamorphosed or "hybridized" into defenses of specialized function. Even without knowledge of the exact chemical nature of diphtheria antitoxin, this conversion of a normal nonspecific defense into a specialized diphtheria antibody might conceivably be effected in the chemical laboratory.

Inspired by this newer concept, Ostromyschlenski and Petroff incubated a mixture of diphtheria toxin and normal horse serum and found, as they had hoped, that the resulting test-tube "conjugates" are specifically antitoxic.[2] During the last two years this observation has been confirmed by Kryshanowski,[3] who was able to synthesize two different diphtheria antitoxins by substituting individual serum proteins for whole serum. By incubating a mixture of diphtheria toxin and commercial trypsin, Shrawosmisslow[4] and Kimmelstiel[5] obtained "toxin-trypsinates," which in their hands were also specifically antitoxic. Artificial diphtheria antitoxin made by a combination of these two methods, *i. e.*, by incubating a mixture of diphtheria toxin and normal horse serum in the presence of commercial pancreatin is at present covered by patent in Germany.[6]

Of course, it is not as yet proved that these synthetic antitoxins are identical with the natural antitoxins formed in the animal body,[5] but there is suggestive evidence that some of them may be even superior to those formed by nature.[2] Successful artificial human convalescent antitoxins made from carefully typed human blood, with the hoped-for elimination of all allergic reactions, is a logical goal for future research.

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1 Manwaring, W. H. A Critique of the Ehrlich Theory, with an Outline of the Enzyme Theory of Antibody Formation—The Newer Knowledge of Bacteriology and Immunology. University of Chicago Press, 1928, chap. 81, p. 1078.

2 Ostromyschlenski and Petroff. Rus. Gesel. f. physical. Chemie, 47, 263, 1915.

3 Kryshanowski, W. H. Centralbl. f. Bakteriol., 110, 1, 1929.

4 Shrawosmisslow, W. H., and Kostromin, N. E. Ztschr. f. Immunitätsforsch, u. exper. Therap., 54, 1, 1927.

5 Kimmelstiel, L. Ztschr. f. Immunitätsforsch u. exper. Therap., 62, 245, 1929.

6 Pat. No. 392055, Class 30 h, Group 6.

Diagnosis of Allergic Rhinitis.—The diagnosis of nasal allergy is based on (1) the history of local symptoms and the local physical findings, (2) the allergic state of the patient. The first requires a thorough knowledge of the standard methods of rhinologic diagnosis and the second, of the principles of immunology.

Eyerman states that there are three methods of investigation on which to base the diagnosis of allergy: (1) an exhaustive and detailed clinical history, considering the presenting symptoms, the family history, the environment and the diet of the patient; (2) sensitization tests properly performed and interpreted; and (3) the demonstration of eosinophils. These are given in the order of their importance.

The acute and seasonal cases with a history of paroxysms of sneezing, obstruction, a watery discharge and edematous mucous membrane, are likely to be confused only with an acute cold. However, the malaise and increased temperature at the onset of a cold followed by a characteristic process of evolution and return to normal in about two weeks is seldom seen in allergic conditions.

When the condition is chronic and hyperplasia of the membrane or polypi are present we must differentiate between an allergic condition and a primary suppuration. The primary suppurations are often unilateral, the edema is limited to the region of the infected cells, and the discharge varies from pure pus to a mixture of pus and mucus. It rarely contains eosinophils. In allergy the lesions are almost always bilateral and involve the whole lining of the nose. The discharge is watery or mucoid and often contains eosinophils. The history alone is often enough to distinguish between the two conditions.

When a combined suppurative and allergic process is present the primary process must be determined by the history. It is probable that most of these are primarily due to allergy and the infection due to the obstruction to ventilation and drainage. This is shown by the number of sinus infections that clear up when allergic conditions are corrected.

The exciting factor may often be determined by the skin tests, or it may be necessary to resort to a process of elimination such as a change in environment, or a study of the diet as has been suggested by Rowe.

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